Individuals identified as psychopathic using Hare’s (1991) Psychopathy Checklist-Revised (PCL-R) are of interest to forensic psychologists because of the high risk that they will engage in antisocial behavior (Hart, 1998). Existing crime data suggest that the PCL-R is a measure with great clinical utility, but evidence concerning the etiology of the PCL-R psychopath is less consistent. We propose that one potential source of the inconsistent evidence is that psychopathy is a construct, like mental retardation, that is etiologically heterogeneous. We suggest that the development of effective clinical interventions will require psychologists to (a) question the assumption that psychopathy is an etiologically homogeneous entity, (b) identify etiologically distinct variants of psychopathy for study, and (c) specify etiological mechanisms that may suggest tangible treatment targets. We discuss two complementary strategies for identifying etiological variants of psychopathy: (a) using general personality theory to identify specific psychopathic traits for study and (b) isolating specific bio-psychological mechanisms that possess the potential to explain specific psychopathic syndromes.

Key words: Psychopathy, Psychopathy Checklist-Revised (PCL-R), psychopathic syndromes. [Clin Psychol Sci Prac 11: 69–94, 2004]

WHAT IS PSYCHOPATHY?
Psychopathy, as it is currently defined, is a personality disorder that manifests early in life and persists...
throughout the life span (Hare, 1996). Psychopathic individuals have traditionally been described as individuals who are lacking in empathy, loyalty, and guilt and who engage in persistent antisocial, impulsive, and irresponsible behavior (Cleckley, 1976). Such individuals typically do not evidence impaired intelligence but appear unable to make use of their intelligence to learn from mistakes (Hart & Hare, 1989). This discrepancy between ability and performance led Cleckley (1976) to conclude that psychopathic individuals suffer from a condition as debilitating as schizophrenia.

Psychopathy is commonly assessed using the Psychopathy Checklist-Revised (PCL-R; Hare, 1991). This 20-item checklist contains both behavioral indicators (e.g., impulsivity) and affective/interpersonal indicators (e.g., shallow affect). Each item is rated zero (not present), one (present to some degree), or two (definitely present). Scores on the instrument thus range from 0 to 40 with scores of 30 or more considered indicative of psychopathic personality (Hare, 1991). Unlike other psychopathy measures that rely only on self-report, PCL-R ratings incorporate collateral information and scores tend to be highly reliable as a result (Hare, 1991). Hare, Hart, and Harpur (1991) have demonstrated that the PCL-R identifies a more specific group that is more predictive of criminal activity than antisocial personality disorder (Hare, 1991). Unlike other psychopathy measures that rely only on self-report, PCL-R ratings incorporate collateral information and scores tend to be highly reliable as a result (Hare, 1991).

Psychopathy and Risk Prediction

Most studies using the PCL-R have focused on the prediction of future risk. Research has demonstrated that male inmates with high PCL-R scores (i.e., PCL-R psychopaths) begin their criminal careers earlier, commit a greater variety of offenses, and offend at higher rates than other inmates (Hare, 1991; Hare & McPherson, 1984; Hart & Hare, 1996; Kosson, Smith, & Newman, 1990; Wong, 1985). Higher PCL-R scores are also associated with higher rates of violent crime (Williamson, Hare, & Wong, 1987), substance abuse (Hart & Hare, 1989; Smith & Newman, 1990), criminal recidivism (Hart, Kropp, & Hare, 1988; Harris, Rice, & Quincey, 1993; Rice & Harris, 1995; Salekin, Rogers, & Sewell, 1996; Serin, Peters, & Barbaree, 1990), and sexual recidivism (Quinsey, Rice, & Harris, 1995; Serin, 1996). Studies using the PCL-R to predict violence have resulted in moderate effect sizes ranging from $r = 0.27$ (Hemphill, Hare, & Wong, 1998) to $r = 0.37$ (Salekin et al., 1996), suggesting that predictions based on psychopathy are significantly better than chance and comparable in accuracy to a variety of important and more commonly accepted psychological, educational, and medical assessments (Hart, 1998). Finally, PCL-R scores have been shown to provide incremental validity in the prediction of violence and recidivism over standard actuarial risk scales based on other demographic and historical variables (Hart et al., 1988).

Predicting behavior, however, is only part of the reason forensic researchers are interested in the assessment of psychopathy. Psychologists are also interested in uncovering the etiological paths that produce the criminal behaviors. Understanding the root causes of antisocial activity is important because it allows us to plan primary prevention and treatment strategies that target key mechanisms. Without an adequate understanding of the underlying etiology, prevention and treatment are likely to be ineffective because they may
target the wrong mechanisms for change. Understanding etiology is also an important prerequisite for exploring the links between behavior and biological mechanisms. Clarification of specific psychological mechanisms makes it more likely that we can identify key biological substrates that might play a part in the development and maintenance of antisocial behavior.

**PSYCHOPATHY: A HOMOGENEOUS ETIOLOGICAL ENTITY?**

Regardless of race, gender, or anxiety level, PCL-R psychopaths show similar levels of criminal versatility, recidivism, and alcohol/drug use (Hart, 1998; Smith & Newman, 1990). This is hardly surprising considering the constellation of traits required to obtain a score of 30 or more on the PCL-R (e.g., lack of remorse, callousness, impulsivity, irresponsibility, deceitfulness, etc.). It is easy to understand why a person who does not feel guilty for past crimes, feel empathy for potential or past victims, or deliberate on the potential costs to himself of his actions might be prone to engage in antisocial behaviors. What remains unclear, however, is whether or not there is a common etiology underlying the expression of these traits in PCL-R psychopaths or if there are different means through which one may come to be considered psychopathic.

Hare (1996) reviewed research indicating that psychopathic individuals demonstrate a variety of laboratory-based indicators of psychopathology. He described a number of well-replicated experimental findings including psychopathic individuals’ reduced skin conductance and startle responses during aversive stimuli (Arnett, 1997; Hare, 1978; Lykken, 1957; Ogloff & Wong, 1990; Patrick, Bradley, & Lang, 1993; Patrick, Cuthbert, & Lang, 1994; Raine & Venables, 1984) as well as their poor passive avoidance (Lykken, 1957; Newman & Kosson, 1986; Newman, Patterson, & Kosson, 1987; Newman & Schmitt, 1998; Thornquist & Zuckerman, 1995). Based on these findings, most agree that the PCL-R does identify a valid construct with important etiological and pathological correlates (Hare, 1996; cf. Harris, Skilling & Rice, 2001).

Unlike the recidivism research, however, there has been a great deal of debate about the implications of these findings. Researchers do not seem able to agree on what the etiological mechanisms that produce psychopathic behavior might be (see Cooke, Forth, & Hare, 1998). Popular suggestions include a lack of normal fearfulness (Lykken, 1995), a cognitive deficit that results in poor response modulation (Newman, 1998), and a more general emotional deficit (Hare, 1998).

In addition, there are data suggesting heterogeneity within the groups identified using the PCL-R. There is, for example, a long history of distinguishing between psychopathic individuals whose behavior appears to be the result of some deficit (“primary” psychopaths) and those whose conduct problems appear to be the result of neurotic conflicts and/or negative affect (“secondary” psychopaths; Blackburn, 1998; Hare, 1970; Karpman, 1941; 1948). Traditionally, such distinctions are made by using measures of fear (e.g., Lykken, 1995) or anxiety such as the Welsh Anxiety Scale (WAS; Welsh, 1956) in conjunction with a measure of psychopathy (Chesno & Kilmann, 1975; Fagen & Lira, 1980; Schmauk, 1970). There is abundant experimental evidence suggesting that anxiety plays an important role in determining whether psychopathic and nonpsychopathic offenders differ on a variety of laboratory measures (Arnett, Howland, Smith, & Newman, 1993; Arnett, Smith, & Newman, 1997; Chesno & Kilmann, 1975; Devonshire, Howard, & Sellars, 1988; Fagan & Lira, 1980; Newman, Patterson, Howland, & Nichols, 1990; Newman, Kosson, & Patterson, 1992; Newman, Schmitt, & Voss, 1997; Schmitt, Brinkley, & Newman, 1999; Smith, Arnett, & Newman, 1992; Widom, 1976).

Empirical evidence relating to the factor structure of the PCL-R also suggests reason to differentiate among the different components or facets of psychopathy. Harpur, Hare, and Hakstan (1989) have demonstrated that the PCL-R consists of two broad and complex factors—one associated with a deviant and antisocial lifestyle and the other associated with the callous and remorseless use of others. Hare (1991) has noted that the two factors have different correlations with a variety of clinical outcome measures. Similarly, Patrick and his colleagues have presented data demonstrating that differences in PCL-R factor scores have important implications for performance on startle tasks (Patrick et al., 1993) and fear conditioning (Patrick et al., 1994). Cooke and Michie (2001) have taken the complexity of the two PCL-R factors even further, suggesting that the PCL-R may in fact be represented better by three factors.
There are also data suggesting that a variety of demographic factors may introduce heterogeneity into PCL-R defined populations. For example, research indicates that the psychopathy construct may not manifest in precisely the same way in Caucasian and African-American samples (Kosson et al., 1990; Lorenz, Bolt, Smith, Schmitt, & Newman, 2000). Similarly, Cooke (1998) has noted that the distribution of PCL-R scores may vary by country. Finally, Moffitt (1993) has argued that individuals demonstrating the same antisocial behavior during adolescence may be delinquent for different reasons (i.e., peer influence vs. possible neuropsychological mechanisms).

Taken together, the existing data suggest that there is significant heterogeneity in PCL-R identified populations. It therefore seems prudent to consider the possibility that there may be multiple causal pathways that result in the constellation of personality traits typically associated with psychopathy (Widiger, 1998).

THE HETEROGENEITY OF PSYCHOPATHY: AN ANALOGY TO INTELLIGENCE

It may seem difficult to reconcile the consistency of the risk-prediction data for the PCL-R with the claim that psychopathy is etiologically heterogeneous. This apparent discrepancy, however, is easily understood in terms of a simple causal chain. Antisocial behavior is best predicted by a collection of traits (impulsivity, callousness, irresponsibility, etc.). The specific constellation of traits involved and the etiological mechanism responsible for those traits, however, may both vary from individual to individual. To illustrate these points, we briefly present a similar consideration of the intelligence construct and discuss its benefits to the study of psychopathy.

Intelligence is generally considered to be a normally distributed dimensional construct, and there are a number of standardized reliable measures designed to assess it (Brody, 2000). When accompanied by deficits in adaptive functioning, global intelligence scores two or more standard deviations below the mean are generally considered indicative of mental retardation (Greenspan, 1999). Mental retardation, in turn, is a reliable predictor of poorer academic performance and economic status in later life (Greenspan, 1999).

Despite its predictive power, the construct of intelligence (as measured by standard tests) does little to define the etiology that produced the low intelligence score in the first place. One can imagine, for example, that some individuals’ scores may be limited as a result of environmental influences on intelligence such as closed-head trauma. Others will have limited intelligence due to discrete biological problems such as phenylketonuria (PKU; Dennis et al., 1999; Waisbren, 1999; Welsh & Pennington, 2000).

Identifying the etiology of various forms of mental retardation is important because of implications for interventions. For example, PKU mental retardation can be treated through diet whereas head-trauma-induced mental retardation may require special rehabilitation therapy (Dennis et al., 1999; Waisbren, 1999; Welsh & Pennington, 2000). Attempts to treat both types of mental retardation in the same manner would be inefficient and ineffective. Thus, knowing and understanding the various manifestations and etiologies underlying mental retardation has little impact on prediction of general performance, but it has important implications for treatment and intervention.

One means of specifying etiological subtypes of mental retardation is to focus on specific components of intelligence. Some researchers have suggested that intelligence is best conceptualized as a collection of multiple processes rather than a single entity (e.g., executive functioning, attention, memory, visual spatial skills, language, etc.; Gardner, 1993; Sternberg, 1985). Using this logic, it is desirable to study specific components of intellectual functioning that may be related to different intellectual limitations. Researchers interested in dyslexia, for example, would want to focus their studies on measures of linguistic intelligence.

Alternately, researchers can search for unitary biopsychological mechanisms that can explain general deficits in intellectual functioning. Sometimes referred to as a taxonomic approach (Gorenstein, 1992), this method requires researchers to (a) develop a theory about a specific process that might disrupt intelligence and (b) determine markers to identify a group of individuals in whom the process operates. Theoretically, individuals who share the markers will show predictable performance differences on measures of intelligence. Down’s Syndrome, for example, is a relatively discrete
syndrome with identifiable markers, and individuals with those markers are known to perform poorly on measures of general intelligence.

Psychopathy can be thought of in a manner similar to the way in which intelligence is conceptualized (Widiger, 1998). Like intelligence, psychopathic personality traits are likely to be distributed along a normal continuum (Lykken, 1995). Some researchers have argued against this point, citing a single study by Harris, Rice, and Quinsey (1994) as evidence for the taxonicity of psychopathy as a whole. This study found support for the taxonicity of Factor 2 items, primarily those dealing with childhood behavior problems, but not for the taxonicity of Factor 1 items (often considered the core of psychopathy). Rather than supporting the simple conclusion that psychopathy is taxonomic, these results suggest that (a) there may be both taxonomic and dimensional elements to psychopathy and (b) the degree of observed taxonomy in the psychopathy construct may be dependent on the measures/methods being used.

Consistent with this view, the manual recommends using the PCL-R to form discrete groups for study (Hare, 1991), but Hare (1996) has also argued that using the measure to study the full range of psychopathic traits is important. This point is underscored by the fact that the full range of PCL-R scores has been found to be predictive of both recidivism (Hart et al., 1988) and diagnoses of DSM personality disorders (Blackburn, 1998). Additionally, studies done using the screening version of the PCL-R (Hart, Cox, & Hare, 1995) with nonincarcerated populations suggest that the relationship between psychopathy and external correlates remains similar even when the overall psychopathy scores are lower than those found in prison samples. Finally, other research examining psychopathy in noninstitutionalized settings suggest that even lower levels of psychopathic traits are predictive of important outcomes (Belmore & Quiney, 1994; Forth, Brown, Hart, & Hare, 1996; Levenson, Kiehl, & Fitzpatrick, 1995; Lilienfeld & Andrews, 1996; Lynam, Whiteside, & Jones, 1999).

Thus, the more psychopathic traits an individual displays, the closer to the psychopathic prototype they are, and the higher their risk for future antisocial activity. Different combinations of traits, however, can produce the same overall PCL-R score and the same risk for antisocial behavior (Hare, 1991). Different etiological mechanisms may be responsible for different combinations of psychopathic traits. There may, therefore, be different etiologies responsible for the same observed psychopathic behaviors.

Any review of the existing psychopathy literature will reveal the presence of numerous, and at times confusingly divergent, theories to account for antisocial behavior (e.g., Dodge, 1993; Hare, 1996; Lykken, 1995; Patrick, 1994; Patterson & Newman, 1993). We suggest that this confusion is due in part to an effort to find a single common explanation for what may in fact represent multiple pathways to common antisocial behaviors. Essentially, we are suggesting that there are a variety of etiological pathways that might produce high scores on the PCL-R. These different etiologic pathways can be relatively independent of risk prediction but not treatment planning. An adequate understanding of the etiology and treatment of psychopathy will require partitioning the different variants of psychopathy in a manner comparable to the identification of the different bases of mental retardation.

IDENTIFYING ETIOLOGICAL VARIANTS OF PSYCHOPATHY

We have, thus far, spent a great deal of time identifying why it may be prudent to consider psychopathy an etiologically heterogeneous entity. We will now turn our consideration to how etiological variants of psychopathy may be identified and studied. We believe that there are at least two different means that may be useful for accomplishing this task.

The first is based in the tradition of Kraepelin (1904), who argued that syndromes would be discovered and understanding advanced through more precise and specific clinical description. In this tradition, we apply a general model of personality functioning that will allow for more specific descriptions of different variants of psychopathy (Lilienfeld, 1994; Lilienfeld & Andrews, 1996; Lykken, 1995; Widiger, 1998; Widiger & Lynam, 1998). Rather than looking for a single etiology for psychopathy as a whole, this approach identifies more specifically the components of personality functioning that lead to a diagnosis of psychopathy. One advantage of this model is its ability to specify individual components of psychopathy for study, which may
prove to be more fruitful than assuming a specific etiology common to all persons who would be diagnosed as psychopathic by the PCL-R. As an example of how general personality theory may contribute to our understanding of psychopathology, we will discuss in detail the use of the Five-Factor Model (FFM) to parse heterogeneity in psychopathy (Widiger & Lynam, 1998).

The second approach, based on the deductive-nomological approach to classification, also flows from the tradition of Kraepelin in that it emphasizes the identification of an underlying etiology that can account for the diverse expression of symptoms found within a syndrome. More specifically, it involves identifying specific psychobiological mechanisms that may be able to account for the range of psychopathic behavior in a specifically defined group of offenders (Bleshfeld, 1984; Cleckley, 1976; Gorenstein, 1992; Newman, 1998; Newman & Brinkley, 1998). In this method, researchers examine whether a theoretically defined diagnostic group differs systematically from controls on measures of the proposed etiological (i.e., causal) processes. The advantage of this approach is that it provides psychologists with clear insight into specific mechanisms with important implications for treatment. To clarify how this approach may be helpful, we will review Newman’s (1998) efforts to elucidate the psychological deficit described by Cleckley (1976).

Method 1: The Five-Factor Model of General Personality Functioning

Widiger and Lynam (1998) have suggested that the FFM may be useful for making further discriminations among PCL-R psychopaths. The FFM was originally developed from studies designed to identify the domains of personality functioning that are most important for describing individual differences (Digman, 1996; Wiggins & Pincus, 1992). The model emphasizes five broad domains of personality: extraversion (positive affectivity), agreeableness, conscientiousness (constraint), neuroticism (negative affectivity), and openness (John & Srivastava, 1999). Based on their research with the NEO Personality Inventory—Revised (NEO PI-R; Costa & McCrae, 1992), Costa and McCrae (1995) have proposed that each of these overarching dimensions can be divided into six facets that can be used to further differentiate individual personality. Table 1 provides a description of the domains and facets of the FFM. Although descriptive, the titles of these domains/facets sometimes fail to capture the full range of the dimensions they represent (Digman, 1990). For example, gullibility, trust, skepticism, cynicism, suspicion, and paranoia represent different points along a continuum of trust versus mistrust, but the full range of the continuum may not be apparent to the reader by the single term of “trust.”

Empirical support for the construct validity of the FFM is extensive, both at the domain and facet levels, including convergent and discriminant validation across self, peer, and spouse ratings (Costa & McCrae, 1988), temporal stability across 7–10 years (Costa, Herbst, McCrae, & Siegler, 2000; Costa & McCrae, 1994), and heritability (Jang, McCrae, Angleitner, Reimann, & Livesley, 1998; Plomin & Caspi, 1999). The FFM has been used as an integrative model of personality functioning for children (Halverson, Kohnstamm, & Martin, 1994), adults (McCrae & Costa, 1990), the elderly (Costa & McCrae, 1994), and even animal species (Gosling & John, 1999).

Studies done in a variety of cultures support the validity of the FFM as a truly general model of personality functioning. Current data suggest that the domains of extraversion, agreeableness, conscientiousness, and neuroticism tend to replicate well across cultures, but openness has received less consistent support (De Raad, Perugini, Hrebiková, & Szarota, 1998; John & Srivastava, 1999; McCrae, 2001; Saucier and Goldberg, 2001). There is also research demonstrating the general cross-cultural applicability of the FFM using both etic (McCrae & Costa, 1997) and emic (Church, 2001; Kagitbik, Church, Guanzon-Lapena, Carlota, & del Pilar, 2002) methodologies.

It should be noted that there are alternative models of personality functioning, including the Big Three model of Tellegen, Clark, and Watson, the seven-factor model of general personality (Tellegen, 1985; Tellegen & Waller, in press), and the interpersonal circumplex model (Benjamin, 1996; Blackburn, 1998; Kiesler, 1996; Wiggins & Pincus, 1992). The differences among these models reflect the difficulties in succinctly defining broad domains of personality (Digman, 1990), variations in belief about the core elements of personality (Ashton,
Lee, & Paunonen, 2002; Lucas, Diener, Grob, Suh, & Shao, 2000), and differing interpretations of the neurobiological substrates of personality (Depue, 1996; Pickering & Gray, 1999). For example, some alternative models have been more explicitly linked with particular physiological systems (i.e., Constraint and the Behavioral Inhibition System; Pickering & Gray, 1999).

Despite these apparent differences, however, there is substantial convergence and commonality between the domains of personality functioning identified by the FFM and the alternative models mentioned above (McCrae & Costa, 1999; Watson & Clark, 1999; Watson & Clark, 1997; Watson, Clark, & Harkness, 1994; Wiggins & Pincus, 1989; Wiggins & Trapnell, 1996). In fact, one of the commendable aspects of the FFM is its ability to represent and account for the major dimensions and components of other dimensional models of general personality functioning (John & Srivastava, 1999; McCrae & Costa, 1999; O’Connor, 2002).

The strong empirical support for the FFM as a model of general personality has led numerous investigators to consider it for use as a dimensional model of personality disorder symptomatology (Clark & Livesley, 2002; O’Connor & Dyce, 1998; Reynolds & Clark, 2001; Widiger & Costa, 1994). In fact, over 50 studies have “examined explicitly the description, classification, or understanding of personality-disorder symptomatology from the perspective of the FFM” (Widiger & Costa, 2002, p. 61). Recent research has demonstrated that (a) DSM-IV personality disorders could be described with good agreement using the FFM, (b) profiles derived from these ratings were similar to those derived empirically and from translations of the DSM criteria, and (c) facet overlap among the FFM profiles repro-

<table>
<thead>
<tr>
<th>Table 1. Domains and Facets of the Five-Factor Model of Personality</th>
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<tr>
<td><strong>Five-Factor Model of Personality Functioning</strong></td>
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<tr>
<td><strong>Neuroticism vs. emotional stability</strong></td>
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<tr>
<td>Anxiousness vs. relaxed, unemotional</td>
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<tr>
<td>Bitter, angry</td>
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<tr>
<td>Optimistic</td>
</tr>
<tr>
<td>Self-consciousness vs. even-tempered</td>
</tr>
<tr>
<td>Tempted, urgency</td>
</tr>
<tr>
<td>Impulsivity vs. controlled, restrained</td>
</tr>
<tr>
<td>Vulnerability vs. stalwart, brave, fearless, unfappable</td>
</tr>
<tr>
<td><strong>Extraversion vs. introversion</strong></td>
</tr>
<tr>
<td>Warmth vs. aloof, indifferent, cold</td>
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<tr>
<td>Gregariousness vs. withdrawn, isolated</td>
</tr>
<tr>
<td>Assertiveness vs. unassuming, quiet, resigned</td>
</tr>
<tr>
<td>Activity vs. passive, lethargic</td>
</tr>
<tr>
<td>Excitement-Seeking vs. cautious, monotonous, dull</td>
</tr>
<tr>
<td>Positive Emotions vs. placid, anhedonic</td>
</tr>
<tr>
<td><strong>Openness vs. closedness to experience</strong></td>
</tr>
<tr>
<td>Fantasy vs. practical, concrete</td>
</tr>
<tr>
<td>Aesthetic vs. unaesthetic</td>
</tr>
<tr>
<td>Feelings vs. unresponsive, constricted, alexithymic</td>
</tr>
<tr>
<td>Actions vs. routine, habitual, stubborn</td>
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<tr>
<td>Ideas vs. pragmatic, rigid</td>
</tr>
<tr>
<td>Values vs. traditional, dogmatic, inflexible</td>
</tr>
<tr>
<td><strong>Agreeableness vs. antagonism</strong></td>
</tr>
<tr>
<td>Trust vs. skeptical, cynical, suspicious, paranoid</td>
</tr>
<tr>
<td>Straightforwardness vs. cunning, manipulative, deceptive</td>
</tr>
<tr>
<td>Altruism vs. selfish, stingy, exploitative</td>
</tr>
<tr>
<td>Compliance vs. oppositional, combative, aggressive</td>
</tr>
<tr>
<td>Modesty vs. confident, arrogant</td>
</tr>
<tr>
<td>Tender-Mindedness vs. callous, ruthless</td>
</tr>
<tr>
<td><strong>Conscientiousness vs. undependability</strong></td>
</tr>
<tr>
<td>Competence vs. lax, negligent</td>
</tr>
<tr>
<td>Order vs. haphazard, disorganized, sloppy</td>
</tr>
<tr>
<td>Dutifulness vs. casual, dependable, unethical</td>
</tr>
<tr>
<td>Achievement-Striving vs. aimless, desultory</td>
</tr>
<tr>
<td>Self-Discipline vs. negligent, hedonistic</td>
</tr>
<tr>
<td>Deliberation vs. careless, hasty, rash</td>
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</table>

Lee, & Paunonen, 2002; Lucas, Diener, Grob, Suh, & Shao, 2000), and differing interpretations of the neurobiological substrates of personality (Depue, 1996; Pickering & Gray, 1999). For example, some alternative models have been more explicitly linked with particular physiological systems (i.e., Constraint and the Behavioral Inhibition System; Pickering & Gray, 1999). Despite these apparent differences, however, there is substantial convergence and commonality between the domains of personality functioning identified by the FFM and the alternative models mentioned above (McCrae & Costa, 1999; Watson & Clark, 1999; Watson & Clark, 1997; Watson, Clark, & Harkness, 1994; Wiggins & Pincus, 1989; Wiggins & Trapnell, 1996). In fact, one of the commendable aspects of the FFM is its ability to represent and account for the major dimensions and components of other dimensional models of general personality functioning (John & Srivastava, 1999; McCrae & Costa, 1999; O’Connor, 2002).

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duced very well the obtained comorbidities among the personality disorders (Lynam & Widiger, 2001). Similar research has also been done with alternative dimensional models of personality functioning (Clark, 1993; Livesley & Jang, 2000). Based on several studies, Clark, Livesley, Schroeder, and Irish (1996) concluded that the higher-order structure of personality disorders could be represented well by four factors common to the five-factor model (neuroticism, introversion, agreeableness, and conscientiousness).

Psychopathy from the Perspective of the FFM. Widiger and Lynam (1998) have proposed that psychopathy can generally be described by a constellation of FFM facets. The FFM conceptualization of psychopathy primarily involves the domains of antagonism (low modesty, straightforwardness, altruism, compliance, and tender-mindedness) and conscientiousness (low dutifulness, deliberation, discipline, and achievement striving).² Some of the complex heterogeneity of psychopathy, however, can be attributed to the presence of other domains and facets of the FFM, including the domains of extraversion (high excitement seeking), high neuroticism (angry hostility and impulsiveness), and low neuroticism (low self-consciousness and low anxiousness).

Careful consideration reveals that all of the core features of psychopathy, as described by Cleckley (1976) and operationalized in Hare’s (1991) PCL-R, have an explicit representation within one or more facets of the FFM. Table 2 provides a translation by Widiger and Lynam (1998) of the PCL-R items into the facets of the FFM. Several aspects of this translation are important to note.

First, facets from the domains of agreeableness and conscientiousness permeate the translation of the PCL-R; facets from agreeableness appear in 14 of the PCL-R items while facets of conscientiousness appear in 13 of the items. For example, the PCL-R item of pathological lying is for the most part a direct expression of FFM low straightforwardness. This facet also represents the PCL-R item “conning/manipulative,” although it is also represented by the facets of altruism and tender-mindedness. Psychopathic callousness can be understood as an expression of the lowest levels of tender-mindedness (see Table 1). Grandiose sense of self-worth is represented by the arrogance and conceit that will occur at the lowest levels of modesty. In terms of the

<table>
<thead>
<tr>
<th>PCL-R Item</th>
<th>FFM Facets</th>
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<tbody>
<tr>
<td>1. Glibness/superficial charm</td>
<td>Low self-consciousness</td>
</tr>
<tr>
<td>2. Grandiose sense of self-worth</td>
<td>Low modesty</td>
</tr>
<tr>
<td>3. Need for stimulation</td>
<td>High excitement-seeking, low self-discipline</td>
</tr>
<tr>
<td>4. Pathological lying</td>
<td>Low straightforwardness, low altruism, low tender-mindedness</td>
</tr>
<tr>
<td>5. Conning/manipulative</td>
<td>Low tender-mindedness</td>
</tr>
<tr>
<td>6. Lack of remorse or guilt</td>
<td>Low warmth, low positive emotionality, low altruism, low tender-mindedness</td>
</tr>
<tr>
<td>7. Shallow affect</td>
<td>Low tender-mindedness</td>
</tr>
<tr>
<td>8. Callous/lack of empathy</td>
<td>Low straightforwardness, low altruism, low modesty, low-tender-mindedness, low achievement striving, low self-discipline</td>
</tr>
<tr>
<td>9. Parasitic lifestyle</td>
<td>High angry hostility, low compliance, low deliberation</td>
</tr>
<tr>
<td>10. Poor behavioral controls</td>
<td>Low straightforwardness, low altruism, low compliance, low modesty, low tender-mindedness, low dutifulness, low self-discipline, low deliberation</td>
</tr>
<tr>
<td>11.Promiscuous sexual behavior</td>
<td>Low straightforwardness, low altruism, low compliance, low self-discipline, low deliberation</td>
</tr>
<tr>
<td>12. Early behavior problems</td>
<td>Low straightforwardness, low altruism, low compliance, low self-discipline, low deliberation</td>
</tr>
<tr>
<td>13. Lack of realistic, long-term goals</td>
<td>Low achievement-striving, low self-discipline</td>
</tr>
<tr>
<td>14. Impulsivity</td>
<td>High impulsiveness, low deliberation</td>
</tr>
<tr>
<td>15. Irresponsibility</td>
<td>Low competence, low dutifulness</td>
</tr>
<tr>
<td>16. Failure to accept responsibility</td>
<td>Low straightforwardness, low tender-mindedness, low dutifulness</td>
</tr>
<tr>
<td>17. Many short marital relationships</td>
<td>Low straightforwardness, low altruism, low compliance, low modesty, low tender-mindedness, low dutifulness, low self-discipline, low deliberation</td>
</tr>
<tr>
<td>18. Juvenile delinquency</td>
<td>Low straightforwardness, low altruism, low compliance, low modesty, low tender-mindedness, low dutifulness, low self-discipline, low deliberation</td>
</tr>
<tr>
<td>19. Revocation of conditional release</td>
<td>Low straightforwardness, low altruism, low compliance, low modesty, low tender-mindedness, low dutifulness, low self-discipline, low deliberation</td>
</tr>
<tr>
<td>20. Criminal versatility</td>
<td>Low straightforwardness, low altruism, low compliance, low modesty, low tender-mindedness, low dutifulness, low self-discipline, low deliberation</td>
</tr>
</tbody>
</table>

Note. Based on Widiger and Lynam (1998).
facets of conscientiousness, dutifulness is a fairly direct indicator of psychopathic irresponsibility. Persons who are abnormally low in dutifulness will be irresponsible, undependable, and unreliable.

Although facets of agreeableness and conscientiousness are the most widely represented, facets from extraversion and neuroticism also appear in the translation. For example, glib and superficial charm is essentially the absence of self-consciousness, a facet of FFM neuroticism (Costa & McCrae, 1995). The average person is characterized by a degree of self-consciousness and will be, to some extent, sensitive to ridicule, prone to embarrassment, socially anxious, awkward, or insecure (Costa & McCrae, 1992; Lykken, 1995). The psychopath, on the other hand, is at the lowest levels of self-consciousness. As described by Cleckley (1941), “more than the average person, he is likely to seem free from social or emotional impediments, from the minor distortions, peculiarities, and awkwardness so common even among the successful” (p. 205).

Although many PCL-R traits are well represented by single FFM facets (e.g., glibness, grandiosity, and lack of remorse) or multiple facets from the same domain (e.g., conning and irresponsibility), other PCL-R traits are represented by combinations of facets from different domains. The degree to which the PCL-R items map onto single FFM domains is, in part, a function of the degree to which the item represents an explicit personality trait rather than a behavior. For example, at least five of the seven PCL-R items translated as combinations of facets from agreeableness and conscientiousness reference antisocial behavior (promiscuous sexual behavior, early behavior problems, juvenile delinquency, revocation of conditional release, and criminal versatility). However, the blended translations offered for these items is quite consistent with what is known about the personality correlates of crime (Miller & Lynam, 2001) and risky sexual behavior (Hoyle, Fejfar, & Miller, 2000). For the remaining PCL-R items the necessity to blend FFM domains was due primarily to either fuzziness in the PCL-R item itself (e.g., shallow affect) or to the placement of related facets, primarily those dealing with impulsivity (see Whiteside & Lynam, 2001), on different domains within the FFM itself (stimulation seeking, poor behavioral controls, and impulsivity).

Consistency of FFM Psychopathy. Research indicates that the FFM translation of the PCL-R provided by Widiger and Lynam is consistent with FFM descriptions of psychopathy obtained by other means. Lynam (2002) provided correlations between the 30 facets of the NEO-PI-R and scores on the Levenson Self-report Psychopathy scale (LSRP; Levenson, Kiehl, & Fitzpatrick, 1995), a scale designed to assess both primary and secondary psychopathy. These correlations were quite consistent with the description provided by Widiger and Lynam. The strongest and most consistent correlations were obtained for the facets of agreeableness and conscientiousness. In fact the correlation between the profile of correlations from Lynam (2002) and the Widiger and Lynam profile is quite high, $r = 0.80$.

Using another approach, Miller, Lynam, Widiger, and Leukefeld (2001) developed a FFM psychopathy prototype from ratings provided by 15 nationally recognized psychopathy researchers on the 30 facets of the FFM. Agreement across the psychopathy researchers was quite good. The average correlation of one rater’s profile description with every other rater’s profile (i.e., inter-rater reliability) ranged from 0.61 to 0.84 with a mean of 0.75. Coefficient alpha for this composite, from an analysis in which raters served as variables and facets as cases, was 0.98.

Miller et al.’s (2001) FFM profile (see Table 3) also agreed well with the PCL-R-based description by Widiger and Lynam (1998). The correlation between them was .77 when the analysis was confined to the facets included by Widiger and Lynam. There were a few notable points of disagreement. For example, whereas the PCL-R description of psychopathy suggested to Widiger and Lynam the presence of low achievement striving (lack of long-term goals) and low competence (irresponsibility), the psychopathy researchers described the prototypic psychopath as being high in competence and achievement striving. The disagreement over competence is likely due to the fact that Miller et al. asked experts to rate how the prototypic psychopath would rate him or herself, whereas Widiger and Lynam judged the competence of the psychopath on the basis of his or her behavior. The disagreement on achievement striving has no easy explanation but was also apparent in expert ratings; this was the most disagreed upon facet in the expert profile.
Researchers also described the prototypic psychopath as being low in FFM anxiousness and vulnerability, two additional traits of psychopathy not represented well by the PCL-R. Cleckley (1976) included in his description of psychopathy an "absence of 'nervousness' or psycho-neurotic manifestations" (p. 206), a component of psychopathy that is represented explicitly by the FFM neuroticism facet of anxiousness (see Tables 1 and 3). "The psychopath is nearly always free from minor reactions popularly regarded as 'neurotic' or as constituting 'nervousness' " (Cleckley, 1976, p. 54).

Miller et al. (2001) correlated the expert consensus FFM psychopathy profile with NEO PI-R self-descriptions provided by 481 young adults participating in a longitudinal study concerning the causes and correlates of substance abuse. The more similar an individual’s NEO PI-R profile was to the FFM prototypic profile for psychopathy, the more he or she was said to have the FFM personality traits associated with psychopathy. The correlation of the individual’s NEO PI-R self-description with the prototype for psychopathy was then used as a measure of psychopathy for each participant.

The NEO PI-R psychopathy index correlated substantially with a self-report psychopathy inventory \( r = 0.46 \); symptoms of antisocial personality disorder \( r = 0.35 \), substance abuse \( r = 0.24 \) for alcohol, and substance dependence \( r = 0.26 \) for alcohol; frequent and varied antisocial activities \( r = -0.39 \); and correlated negatively with internalizing symptoms of anxiety \( r = -0.07 \) and depression \( r = -0.18 \). All of the findings replicated in magnitude the results previously reported using incarcerated, PCL-R-defined psychopaths. The ability of the NEO PI-R psychopathy index to match the results obtained by the PCL-R is particularly impressive, given that the NEO PI-R does not include any items concerned explicitly with the criminal, drug, exploitative, and other psychopathic behaviors that are a major component of the PCL-R assessment. Further empirical support for a FFM conceptualization of psychopathy has been reported by Harpur, Hart, and Hare (1994), Hart and Hare (1994), Lynam et al. (1999), and Miller and Lynam (in press).

**Implications of FFM Psychopathy.** There are several advantages of using the FFM to parse the heterogeneity of psychopathy. The FFM is a well-regarded and well-understood model of personality with a large body of supportive research (Digman, 1996; John & Srivastava, 1999). To the extent that psychopathy can be understood in terms of the traits within the FFM, a substantial amount of basic research on general personality functioning can be easily brought to bear on our understanding of the etiology of many psychopathic behaviors. For example, research on the structure (John & Srivastava, 1999), genetics (Plomin & Caspi, 1999), neurobiology (Depue & Lenzenweger, 2001), and development (Caspi, 1997) of personality can be applied to research on psychopathy to generate theory and to extend our understanding of mechanisms and treatment.

PCL-R psychopathy has been compared favorably with the antisocial personality disorder diagnosis (APA,
1994) because it places more emphasis on the assessment of personality traits than on criminal and delinquent behaviors (Hare et al., 1991; Widiger & Corbitt, 1995). However, many of the PCL-R items also lack specific referents (Lilienfeld, 1994). For example, juvenile delinquency and revocation of conditional release are behavioral indicators of psychopathy but they do not themselves refer to or identify a particular personality trait. As suggested by the FFM conceptualization of the PCL-R items (see Table 2), several PCL-R items can result from a variety of personality traits. For example, revocation of a conditional release could be the result of low straightforwardness (deception, manipulation), low altruism (exploitation), low compliance, low dutifulness, low self-discipline, or low deliberation. Individuals who fail on probation because of poor self-discipline are theoretically different from those who fail because of low dutifulness. A FFM description may, therefore, enhance our understanding of the personality traits that drive psychopathic behaviors and provide helpful ideas for structuring successful interventions.

Perhaps most importantly, the FFM understanding of psychopathy may hold the key to resolving several lingering issues in the field, including the two-factor structure of the PCL-R, the variety of conceptions of successful psychopathy, and the diversity of psychopathic deficits. Each of these issues will be discussed in turn.

Although several studies have identified a two-factor structure underlying PCL-R scores, the interpretation of these factors has been unsatisfactory (Cooke & Michie, 2001; Lilienfeld, 1994; Rogers & Bagby, 1994). The first factor is traditionally identified as “a constellation of interpersonal and affective traits commonly considered to be fundamental to the construct of psychopathy” whereas the second factor reflects a “chronically unstable, antisocial, and socially deviant lifestyle” (Hare, 1991, p. 38). This interpretation has been criticized for failing to provide a meaningful conceptualization of the second factor, as if this factor has little to do with being psychopathic (Lilienfeld, 1994).

Emphasis has often been placed on the first factor relative to the second, despite the fact that the second factor includes aspects of personality functioning (e.g., impulsivity, irresponsibility, and sensation-seeking) that could be equally important to the etiology and pathology of the disorder (Rogers & Bagby, 1994; Widiger & Lynam, 1998). “Both PCL factors represent personality traits” (Lilienfeld, 1994, p. 28). In fact, if one takes the FFM facet translations of the PCL-R items presented in Table 2, identifies the higher-order FFM factors in which they are placed, and organizes them with respect to the Harpur et al. two-factor structure, considerable clarity and understanding of the personality functioning represented by the two factors does emerge. The FFM conceptualization of the two-factor structure is presented in Table 4.

The constellation of interpersonal and affective traits that form the first PCL-R factor are primarily facets of FFM antagonism. The unstable, antisocial, and deviant lifestyle factor is a mixture of facets of low conscientiousness and antagonism. The first factor has been more readily characterized in the literature because it is confined largely to one domain of the FFM, antagonism. The second factor has been more difficult to discern conceptually because it includes traits that are suggestive

<p>| Table 4. The Harpur et al. (1988) Two PCL-R Factors from the Perspective of the FFM |
|------------------------------------------|-------------------------------|</p>
<table>
<thead>
<tr>
<th>PCL-R Item</th>
<th>FFM Domain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Factor 1</strong></td>
<td></td>
</tr>
<tr>
<td>1. Glibness/superficial charm</td>
<td>Low neuroticism</td>
</tr>
<tr>
<td>2. Grandiose sense of self worth</td>
<td>High antagonism</td>
</tr>
<tr>
<td>4. Pathological lying</td>
<td>High antagonism</td>
</tr>
<tr>
<td>5. Conning/manipulative</td>
<td>High antagonism</td>
</tr>
<tr>
<td>6. Lack of remorse or guilt</td>
<td>High antagonism, low extraversion</td>
</tr>
<tr>
<td>7. Shallow affect</td>
<td>High antagonism</td>
</tr>
<tr>
<td>8. Callous/lack of empathy</td>
<td>High antagonism</td>
</tr>
<tr>
<td>16. Failure to accept responsibility</td>
<td>High antagonism, low conscientiousness</td>
</tr>
<tr>
<td><strong>Factor 2</strong></td>
<td></td>
</tr>
<tr>
<td>3. Need for stimulation</td>
<td>Low conscientiousness, high extraversion</td>
</tr>
<tr>
<td>9. Parasitic lifestyle</td>
<td>Low conscientiousness, high antagonism</td>
</tr>
<tr>
<td>12. Early behavior problems</td>
<td>Low conscientiousness, high antagonism</td>
</tr>
<tr>
<td>13. Lack of realistic, long term goals</td>
<td>Low conscientiousness</td>
</tr>
<tr>
<td>14. Impulsivity</td>
<td>Low conscientiousness, high neuroticism</td>
</tr>
<tr>
<td>15. Irresponsibility</td>
<td>Low conscientiousness</td>
</tr>
<tr>
<td>18. Juvenile delinquency</td>
<td>Low conscientiousness, high antagonism</td>
</tr>
<tr>
<td>19. Revocation of conditional release</td>
<td>Low conscientiousness, high antagonism</td>
</tr>
<tr>
<td>10. Poor behavioral controls</td>
<td>Low conscientiousness, high antagonism, high neuroticism</td>
</tr>
</tbody>
</table>

Note. Based on Widiger and Lynam (1998).
of two different domains of the FFM, low conscientiousness and antagonism. Nevertheless, there are as many elements of personality within the second factor as the first that contribute to the development of the deviant and unstable antisocial lifestyle, such as low dutifulness, low self-discipline, low deliberation, low compliance, high impulsivity, high excitement-seeking, and low altruism (Lilienfeld, 1994; Rogers & Bagby, 1994). Additionally, the FFM interpretation of the two factors explains well the substantial correlation of 0.50 that is commonly obtained between the two factors (Hare, 1991), as both factors include facets of antagonism (Lynam, 2002).

The FFM also brings clarity to the variety of conceptions of “successful” psychopathy. Cleckley (1976) described “incomplete manifestations of the disorder” (p. 188) by which he meant to refer to individuals in whom the “psychopathologic process . . . has not crowded ordinary successful functioning in the outer aspects of work and social relations entirely out of the picture” (p. 189). Various case studies describe businessmen, scientists, physicians, and other individuals who demonstrate many psychopathic characteristics but manage to function successfully in the community and avoid incarceration (Cleckley, 1976; Hare, 1993). These individuals are difficult to identify for study since traditional psychopathy assessments (DSM-IV, PCL-R, etc.) rely to some degree on antisocial behavior as a criteria. The FFM conceptualization, however, relies only on personality traits. It suggests that successful psychopaths can be identified by looking for individuals who meet a particular subset of the traits in the FFM psychopathic profile. The individuals that Cleckley and Hare described are clearly deceptive, exploitive, arrogant, and callous (i.e., high in antagonism). However, these individuals have frequently obtained advanced degrees and moved far in their fields. They would seem to lack other important characteristics possessed by the prototypic psychopath such as unreliability, aimlessness, and poor impulse control (i.e., low conscientiousness). The FFM profile, then, can be used to create assessments that would be useful for studying “successful” psychopaths.

The FFM can also be used to systematically study proposed differences between “successful psychopaths” and “heroes,” whom some have seen as closely related. For example, Lykken (1995) has written “that the hero and the psychopath are twigs from the same branch. Both are relatively fearless.” This description, however, focuses only on one aspect of the FFM profile for psychopathy (low neuroticism). Where the two profiles should differ is in terms of their levels of antagonism (i.e. deceptiveness, aggressiveness, etc.) and conscientiousness (i.e. reliability, dedication, etc.). In short, the hero may share the characteristic of low fear with the psychopath, but there are more elements to prototypic psychopathy than low fear.

Within the FFM, understanding temperament is important to explaining behavior. As such, differences in personality profiles are potentially important indicators of differing etiological processes. The FFM, then, can be used to generate and test hypotheses about etiological processes that may underpin the specific psychopathy-related facets of the NEO PI-R (Lynam, 2002; Widiger, 1998). This may be accomplished by identifying groups that differ on only one key aspect of the FFM psychopathy profile. Researchers could then determine what kinds of experimental correlates are specifically associated with the key dimension.

Patrick et al. (1993) have reported an emotional hyporeactivity in psychopathic individuals as assessed by a fear-potentiated startle response. Patrick (1994) has specifically related this deficit to the broad domain of negative affectivity (neuroticism) of the FFM. Citing the research of Cook, Stevenson, and Hawk (1993), he indicated that “subjects high in negative affectivity [neuroticism] showed dramatic startle potentiation during unpleasant imagery whereas low negative emotionality subjects showed no such effect” (p. 324). Drawing a connection between temperament and pathology, Patrick (1994) suggested “the observed absence of startle potentiation in psychopaths (Patrick et al., 1993) may reflect a temperamental deficit in the capacity for negative affect” (Patrick, 1994, p. 325). A researcher testing Patrick’s (1994) hypotheses concerning abnormal affective response could use the PCL-R and NEO PI-R to identify subsets of psychopathic individuals who differ in their levels of neuroticism and then administer experimental measures of emotional processing (such as affect-modulated startle reflex) to evaluate the degree to which task performance varies with levels of neuroticism.

Lykken (1995) suggested that the etiology of psychopathy may be constitutionally low levels of fear.
In support of this hypothesis, Lykken (1995) cited data suggesting that psychopathic individuals demonstrate lower skin conductance (SC) when faced with negative stimuli and are more likely than controls to indicate a preference for risky rather than boring activities on the Activities Preference Questionnaire (APQ). Although it has been argued that neither SC nor the APQ is a pure measure of FFM neuroticism (Fowles, 2000; Lykken, 1995), the constructs are related—particularly to the FFM facet of vulnerability that is another measure of fearfulness (Costa & McCrae, 1995). In the context of the FFM, Lykken’s (1995) hypothesis could be tested by identifying participants with low levels of vulnerability and determining whether or not they show the behaviors and experimental performance expected from psychopathic individuals.

Newman (1998) has proposed that the psychopath has a deficit in the ability to suspend a dominant response set in order to assimilate feedback from the environment and/or shifting attention from the organization/implementation of behavior to its evaluation. The FFM describes this pathology as excessively low conscientiousness, particularly the facets of deliberation and discipline. Persons low in discipline are impaired in their “ability to begin tasks and carry them through to completion despite boredom and other distractions,” and persons low in deliberation are “hasty and often speak or act without considering the consequences” (Costa & McCrae, 1992, p. 18). The description of the attentional deficit by Newman is clearly more specific and precise than the description of discipline and deliberation by Costa and McCrae (1992), but “a lack of prospective reflection or, in other words, a lack of planful thought and sound judgment” (Patterson & Newman, 1993, p. 722) is likely to be related in meaningful ways to deficits in deliberation, planfulness, and discipline that are evident in persons who are excessively low in conscientiousness. Tests of this hypothesis might be accomplished using the FFM by identifying psychopathic participants who vary in their levels of deliberation and/or discipline to see if they behave distinctively in laboratory situations.

Personality, like intelligence, is a broad, multifactorial construct. It includes many varied, but correlated, components of functioning that will have resulted from a variety of complexly interacting etiologies. One strength of the FFM conceptualization of psychopathy is its recognition of this complexity and its avoidance of overly simplified models of etiology. “In approximately 30–40% of individuals seen in clinical settings, no clear etiology for . . . Mental Retardation can be determined” (APA, 1994, p. 43). This is, in part, because the level of intelligence of most individuals, including many of the mentally retarded, will have been the result of a complex array of multiple genetic, fetal and infant development, and environmental influences. Mental retardation is a mental disorder for which “there are more than 200 recognized biological syndromes . . . entailing disruptions in virtually any sector of brain biochemical or physiological functioning” (Popper & Steingard, 1994, p. 777). The same can be said for personality functioning and for the etiology of maladaptive personality traits.

The maladaptive personality traits evident within persons who meet the PCL-R criteria for psychopathy may also be the result of a variety of complexly interacting etiologies. All instances of maladaptively low levels of conscientiousness and high levels of antagonism are unlikely to be the result of one specific etiology. There are likely to be multiple pathways toward developing low conscientiousness, just as there are multiple etiologies for maladaptively low levels of intelligence. However, some instances of maladaptive low conscientiousness could be the result of a specific etiology and pathology, such as deficits in response modulation.

Method 2: The Search for Unifying Bio-Psychological Mechanisms (The Deductive-Nomological Approach)

Although there are many cases of mental retardation that have no specific etiology, there are also instances that can be traced to a specific etiology and pathology (e.g., Down syndrome resulting from trisomy 21). In such cases, a discrete dysfunction is found to adversely affect a variety of intellectual processes that, combined, produce a distinctive mental retardation syndrome. The identification and isolation of these specific variants of mental retardation obviously have major implications for diagnosis and treatment. There may well be cases similar to Down syndrome where a single etiology is capable of explaining the dysfunctional behavior of a subgroup of psychopathic individuals. One means of advancing our understanding of psychopathy is to isolate these theoretically homogeneous variants and
identify the bio-psychological mechanisms associated with them (Blashfeld, 1984; Gorenstein, 1992), an approach similar to that advocated by Cleckley (1976).

Investigators using this approach typically begin by identifying individuals displaying a distinctive combination of symptoms, referred to as a clinical syndrome. In this method, individual traits or symptoms are generally not considered particularly important and may vary from individual to individual. Symptoms are of interest because particular combinations of them are believed to reflect larger, independent processes with potential etiological significance. For example, fever, nausea, and loss of appetite may be understood as three distinct processes. Their co-occurrence, however, sometimes reflects an independent disease process that must be understood in its own right (i.e., influenza). So, too, we propose that the behavioral traits manifested by some psychopaths are best understood as diverse and variable manifestations of a psychobiological process that is distinct from those that normally underlie the specific traits.

The next step is to postulate a specific bio-psychological mechanism that may underlie the clinical syndrome of interest. This mechanism, in essence, defines a set of interconnected laws, or a nomological net (Cronbach & Meehl, 1955), that links observable behaviors to unobservable processes. Progress in understanding the syndrome occurs by investigating testable hypotheses regarding these links. In this way, the taxonomic category begins to stand for a theoretical process that explains the syndrome as opposed to a list of symptoms.

In the event that particular hypotheses are not supported by the research, investigators explore whether the nomological network requires revision or the method for classifying individuals needs refinement. The ultimate goal of this “bootstrapping” process is to identify a taxonomic category that signifies a lawful set of relationships capable of explaining the relevant behavior. In theory, this process continues until researchers are able to provide clinicians with measures that can reliably identify the specific deficits in question and suggestions about how the pathology might be treated.

One criticism of this approach is that not all psychopathology can be cleanly described in terms of discrete syndromes with unitary causes (Millon, 1991). This approach can, however, provide us with an excellent tool for researching the cases like Down syndrome that can be described in discrete terms (Gorenstein, 1992). Research into relatively discrete bio-psychological mechanisms has already contributed significantly to our understanding of the pathology associated with psychopathy (Hare, 1998; Newman, 1998). A specific benefit to the taxonomic approach is that once a discrete etiological mechanism is identified, treatment programs can be developed that address the individual problems caused by the specified mechanism (Gorenstein, 1992; Skinner, 1981). In fact, if markers for the syndrome can be identified, eventually it may be possible to intervene proactively before problematic behaviors manifest.

The following sections will focus on a specific etiological mechanism, originally proposed by Cleckley (1976), that Newman and Brinkley (1998) have proposed has important implications for the understanding and treatment of psychopathy. These sections will address traditional means used to identify individuals believed to possess the deficit and will review examples of the empirical evidence for this etiology. It is hoped that this will serve as an example of how the deductive nomological approach to taxonomic classification may continue to help improve our theoretical and practical understanding of psychopathy.

Cleckley’s Pathology and the Response-Modulation Hypothesis. Cleckley (1976) proposed the existence of a distinct group of psychopathic individuals whose poorly regulated behavior could be explained by a discrete pathology. More specifically, he argued that the failure of psychopathic individuals to use their good intelligence to control behavior, to learn from mistakes, or to demonstrate appropriate affect might be due to “a serious and subtle abnormality or defect at deep levels disturbing the integration and normal appreciation of experience” (p. 388). He also stated that his “concept of the psychopath’s functioning postulates a selective defect or elimination which prevents important components of normal experience from being integrated into the whole human reaction” (p. 374).

In his efforts to specify the deficit in question, Cleckley (1976) suggested that psychopathic individuals have difficulty putting conceptual understandings into practice. Concerning the psychopathic individual, he
wrote: “So long as the test is verbal or otherwise abstract, so long as he is not a direct participant, he shows he knows his way about. . . . When the test of action comes to him we soon find ample evidence of his deficiency” (Cleckley, 1976; p. 346). Thus, it has been argued that Cleckley’s (1976) proposition was that psychopathic individuals have a cognitive/affective deficiency that impairs their ability to utilize their knowledge and experience to regulate ongoing behavior (Hare, Williamson, & Harpur, 1988; Newman, 1998; Newman & Brinkley, 1998; Newman & Wallace, 1993).

Although researchers were intrigued by Cleckley’s (1976) hypothesis, they initially had great difficulty determining what the mechanism in question might be and how it might be measured (Lykken, 1957). Since his original proposal, however, there has been a virtual renaissance in cognitive (Posner, 1989) and affective neuroscience (Davidson, 1992) that has provided psychology with more sophisticated and reliable methods of measuring cognitive/affective processing (Newman, 1998). This has made it possible for researchers to consider more carefully Cleckley’s ideas concerning the pathology of the psychopathic individual.

Based in part on these observations by Cleckley (1976), Newman (1998) has developed a theory concerning one possible etiology of psychopathy—the response-modulation hypothesis. Within this proposal, response modulation is defined as a brief and relatively automatic shift of attention from the effortful organization and implementation of goal-directed action to accommodate secondary/peripheral (i.e., latent) information. Note that there are three key components to this definition.

First, for response modulation to be relevant, an individual must have his or her attention committed to an ongoing, goal-directed activity. Second, the shift of attention between primary and secondary cues needs to be relatively automatic as opposed to deliberate. Third, the secondary or peripheral information must be potentially relevant to task performance. Since secondary/peripheral information is defined by the absence of deliberate attention, there are a variety of different kinds of cues that might qualify. Examples include stimuli that are spatially peripheral to a person’s primary focus of attention (e.g., Newman et al., 1997), consideration of past or changing consequences associated with responses (e.g., Newman et al., 1987, 1990), and emotional connotations as well as other (e.g., semantic) aspects of target stimuli that are not the focus of deliberate processing (Lorenz & Newman, 2002; Williamson, Harpur, & Hare, 1991).

Newman (1998) has suggested that the cognitive/affective deficit discussed by Cleckley (1976) may well be poor response modulation. According to Newman and Wallace (1993), deficient response modulation may provide a ready explanation for the core characteristics of psychopathy described by Cleckley (1976; see also Newman & Lorenz, in press).

Identifying Etiologically Homogeneous Groups. Evaluating specific etiological hypotheses about the nature of psychopathy requires identification of diagnostic indicators that presumably signify the nomological net. Cleckley (1976) attempted to accomplish this by compiling a list of behaviors he believed to be diagnostic indicators of his proposed bio-psychological mechanism. This list included such traits as superficial charm, unreliability, insincerity, callousness, egocentricity, and a failure to learn from experience. Individually, none of these traits is particularly discriminating and, in fact, might be commonly found amongst a variety of antisocial individuals. The logic, however, is that an individual possessing a critical mass of these indicators is likely to possess the syndrome/etiology of interest (Blashfeld, 1984; Gorenstein, 1992).

Although the inclusion criteria are helpful for identifying individuals with the proposed syndrome, Cleckley (1976) also argued that it would be necessary to distinguish psychopathy from alternative syndromes with superficially similar antisocial outcomes. Cleckley considered this task so important that he devoted a 72-page section of the Mask of Sanity to distinguishing psychopathic individuals from a variety of other groups whom he believed shared similar traits, but different etiologies (i.e., ordinary criminals, mental defectives, hedonists, etc.). In particular, he wanted to distinguish his construct from antisocial behavior that was driven by psychosis, “psychoneurotic manifestations,” and poor intelligence. To ensure that such factors did not cloud any scientific observations, he also proposed exclusion criteria that could be used to help identify the clearest cases of his proposed syndrome—absence of
“nervousness,” absence of delusions/irrational thinking, absence of low intelligence. In fact, Cleckley believed these exclusion criteria to be so important that he included them as the first 3 of his 16 core characteristics of psychopathy.

The PCL-R is sometimes thought to be a measure of Cleckley’s construct. This is an understandable assumption since the instrument was originally designed with that intent (Hare, 1980). In fact, many of the individual items correspond closely to Cleckley’s inclusion criteria (Hare, 1991). As such, the PCL-R provides an excellent starting point for researchers wishing to study the etiology proposed by Cleckley. It should be noted, however, that Cleckley’s exclusion criteria are not represented by items on the PCL-R (i.e., absences of “nervousness,” “good intelligence,” and “absence of delusions”; Hare, 1991; Schmitt & Newman, 1999). As a result, the “syndrome” we are attempting to examine may be obscured by factors such as anxiety, poor intelligence, or psychosis when using the PCL-R alone.

Following this logic, Newman (1998) has argued that researchers wishing to study hypotheses based on Cleckley’s work should make use of additional measures to refine diagnoses made using the PCL-R. Traditionally, Newman (1998) has used anxiety and intelligence measures to assess Cleckley’s (1976) exclusion criteria. Similarly, psychotic subjects have been eliminated because Cleckley (1976) stated that his participants had an “absence of delusions.” By excluding individuals with psychoses, high anxiety, and low intelligence from our psychopathic syndrome, we are not suggesting that the excluded individuals represent a second distinct diagnostic category (e.g., secondary psychopaths). Rather, the use of these exclusion criteria is intended to create a more homogenous diagnostic group by removing the effects of alternative etiological processes that may co-occur with the etiology under consideration.

For example, neurotic anxiety has been discussed as a potential etiology for persistent antisocial behavior, but this etiology is distinct from the etiological processes underlying primary psychopathy (Cleckley, 1976; Hare, 1970; Karpman, 1941). In light of the fact that anxiety and psychopathy are independent (Schmitt & Newman, 1999), both processes can co-occur in individuals selected for study. Individuals with low anxiety can display similar deficits to those diagnosed with psychopathy. For example, low-anxious individuals’ lack of reactivity to threat cues results in lower electrodermal activity, like that displayed by psychopathic individuals (Arnett, 1997). Thus, without controlling for anxiety it is not possible to know which etiological process accounts for observed differences on experimental measures. We therefore want to maximize our opportunities to observe the systematic effects of the proposed etiological mechanism unhindered by potentially confounding influences.

**Testing the Pathological Mechanism.** The next step is to see whether or not the identified individuals demonstrate the types of deficits associated with the proposed pathology. This step has important implications, not only in verifying the presence of the bio-psychological mechanism, but also in clarifying how the mechanism operates. It is through this basic research that psychologists are able to identify the deficits of psychopathic individuals and the precise conditions under which they manifest. In the interests of brevity, only a brief review of the evidence supporting the response modulation is presented here (see Newman, 1998 and Newman & Lorenz, in press).

Newman (1998) has predicted that psychopathic individuals meeting Cleckley’s diagnostic and exclusion criteria will show performance discrepancies on tasks requiring response modulation and that their poor response modulation will distinguish them from controls who are similarly free from the potentially obscuring factors of psychosis/anxiety and poor intelligence. Consistent with these predictions, various studies have demonstrated that low-anxious psychopathic individuals (but not low-anxious controls) demonstrate deficient performance when they are placed in situations requiring them to alter a dominant response set for reward (Newman et al., 1990, experiment 1; Newman et al., 1992; Newman & Schmitt, 1998). For example, Newman and Schmitt (1998) presented participants with a go/no-go discrimination (passive avoidance) task. Participants were told they would observe a set of two-digit numbers and their goal was to respond to “good” numbers and not respond to “bad” numbers. Early trials were organized to encourage a dominant response set of responding for reward. Thus, successful performance required participants to suspend
their dominant response set in order to process negative feedback and inhibit responses in the presence of punishment stimuli. As predicted, low-anxious psychopathic individuals were less likely to utilize feedback from punished responses to revise future responses and demonstrated poor passive avoidance performance (see Table 5).

Newman’s (1998) theory also suggests that psychopathic individuals will fail to benefit from information that is peripheral to their deliberate focus of attention but potentially helpful for task performance (Lorenz & Newman, 2001; Newman et al., 1997; Newman & Lorenz, in press; W. A. Schmitt, unpublished). In a lexical-decision task, for example, the focus of the task is deciding whether or not a string of letters spells a word. When the target is a word, it possesses incidental information (semantic meaning, emotionality, frequency of use, etc.) that is not necessary for making lexical decisions but which may facilitate task performance. The response-modulation hypothesis predicts that controls, but not psychopathic individuals, will automatically make use of these incidental aspects while making lexical decisions. Consistent with this prediction, Lorenz and Newman (2002) found that controls were faster to identify emotional and high-frequency words than affectively neutral and low-frequency words, whereas psychopathic individuals were relatively unaffected by the words’ affective connotations or frequency (see Table 5).

The response-modulation hypothesis also outlines the conditions under which psychopathic individuals demonstrate performance deficits. For example; Cleckley psychopaths should demonstrate deficiencies in situations in which they are required to alter an ongoing response set or focus of attention. Consistent with this prediction, low-anxious psychopathic individuals have been found to perform like normals in tasks that do not require them to alter a dominant response set (Newman et al., 1990, experiment 3) or process peripheral stimuli (W. A. Schmitt, unpublished).

Newman et al. (1990, experiment 3) administered a passive avoidance task in which participants viewed pairs of numbers and decided whether a given number was “good” (i.e., responding results in reward), “bad” (i.e., responding results in punishment), or “neutral” (i.e., responding results in neither reward nor punishment). One number in each pair was “neutral” and the other was either “good” or “bad.” Unlike the Newman and Schmitt (1998) task, participants had to respond on each trial. Participants were told to respond to “good” numbers when they appeared as part of a pair and respond to “neutral” numbers when “bad” numbers appeared as part of a pair. Thus, task instructions encouraged participants to adopt a response set that

Table 5. Experimental Evidence for Hypotheses Relating to the Cleckley Psychopathic Subtype

<table>
<thead>
<tr>
<th>Study</th>
<th>Paradigm</th>
<th>Model prediction</th>
<th>Low-anxious psychopathic data</th>
<th>Low-anxious control data</th>
<th>Significant differences reported?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newman &amp; Schmitt (1998)</td>
<td>Go/no-go task (10 stimuli)</td>
<td>Low-OWAS PP will show poor PA performance</td>
<td>Mean PA errors = 22.6</td>
<td>Mean PA errors = 16.4</td>
<td>Yes</td>
</tr>
<tr>
<td>Newman et al. (1990)</td>
<td>Go/no-go task (8 stimuli)</td>
<td>Low-OWAS PP will show poor PA performance</td>
<td>Mean PA errors = 14.2</td>
<td>Mean PA errors = 8.79</td>
<td>Yes</td>
</tr>
<tr>
<td>Arnett et al. (1993)</td>
<td>Go/no-go task with long ISI</td>
<td>No Prediction</td>
<td>Mean PA errors = 8.0</td>
<td>Mean PA errors = 10.6</td>
<td>No</td>
</tr>
<tr>
<td>Newman et al. (1990)</td>
<td>Classify good vs. bad numbers</td>
<td>No group differences</td>
<td>Mean classification error = 13.5</td>
<td>Mean classification error = 14.7</td>
<td>No</td>
</tr>
<tr>
<td>Newman et al. (1997)</td>
<td>Picture-word task</td>
<td>Low-OWAS PP will show less Interference from peripheral stimuli</td>
<td>Mean Interference = –18 ms</td>
<td>Mean interference = 58 ms</td>
<td>Yes</td>
</tr>
<tr>
<td>Lorenz &amp; Newman (2001)</td>
<td>Lexical Decision task</td>
<td>Low-OWAS PP will show less emotional facilitation</td>
<td>Mean facilitation = 7.7 ms</td>
<td>Mean facilitation = 33.29 ms</td>
<td>Yes</td>
</tr>
<tr>
<td>Lorenz &amp; Newman (2001)</td>
<td>Lexical Decision task</td>
<td>Low-OWAS PP will show less facilitation from frequency of word use</td>
<td>Mean facilitation = 18.37 ms</td>
<td>Mean facilitation = 51.82 ms</td>
<td>Yes</td>
</tr>
</tbody>
</table>

WAS = Welsh Anxiety Scale; PP = Psychopathic Participants; PA = Passive Avoidance.
involved processing both punishment and reward cues. Under these conditions, low-anxious psychopathic individuals performed as well as low-anxious controls on both the reward seeking and avoidance component of the task (see Table 5). Psychopathic individuals have also been shown to perform as well as controls on tasks that encourage a single dominant response by relying solely on either reward feedback or punishment feedback (Newman & Kosson, 1986).

The response-modulation hypothesis also predicts that these deficits are specific to situations in which the processing of the contextual information is relatively automatic. Studies that have used longer inter-stimulus intervals to provide participants with additional time to process feedback and/or evaluate their strategy have found that low-anxious psychopathic individuals do not demonstrate performance discrepancies. For example, Arnett et al. (1993) administered a version of the go/no-go discrimination task used by Newman & Schmitt (1998). The version administered by Newman and Schmitt used a fixed 1-s inter-stimulus interval whereas the version administered by Arnett et al. used a random inter-stimulus interval lasting between 8 and 14 s. Low-anxious psychopathic individuals demonstrated poor passive avoidance in the Newman and Schmitt (1998) study but performed as well as controls in the Arnett et al. study (see Table 5).

Finally, the response-modulation hypothesis predicts that low-anxious psychopaths will have difficulty processing affectively neutral as well as affectively significant peripheral stimuli in situations requiring response modulation. Consistent with this prediction, low-anxious psychopathic individuals demonstrated performance deficiencies on tasks involving emotionally neutral stimuli (Newman et al., 1997; W. A. Schmitt, unpublished). For example, Newman et al. (1997) examined Stroop-like interference using picture-word tasks. As expected, nonpsychopathic participants were slower to name or make judgments about the pictures and words when they appeared with a peripheral stimulus that was incongruent with the target stimulus. Even though the peripheral pictures and words used in the study were affectively neutral, the incongruent peripheral information engendered significantly less interference in psychopaths than in controls.

In summary, extant research appears consistent with the idea that low-anxious psychopathic individuals demonstrate performance deficits that distinguish them from low-anxious controls. Further, the specific deficiencies and conditions under which they are manifested are consistent with the proposed etiological mechanism of poor response modulation. Of particular note are the findings that suggest that (a) psychopathic individuals are deficient in the use of peripheral cues, (b) this deficit is relatively specific to circumstances requiring automatic shifts of attention, (c) the deficit applies to a range of potentially important incidental cues, and (d) the deficit may underlie psychopathic individuals' specific failure to inhibit punished responses and more general difficulty "accommodating the affective components of experience." The preliminary data, then, seem encouraging enough to warrant further consideration of poor response modulation as the etiology for a subgroup of PCL-R psychopaths meeting Cleckley's criteria.

**Relationship to Antisocial Behavior.** Recall that an important goal of specifying an etiologically homogeneous group of psychopaths is to gain a better understanding of the mechanisms that drive individuals to engage in persistent, self-destructive, antisocial behavior. Although PCL-R psychopaths are at high risk to engage in these sorts of behaviors (Hare, 1996), there is no guarantee that a subgroup of them will show the same high risk for antisocial behaviors. Specifying a potentially etiologically homogeneous group of PCL-R psychopaths is less useful if the identified individuals don’t engage in the same high-risk behaviors researchers are interested in understanding. It is important, then, to consider if low-anxious psychopathic individuals demonstrate the same high-risk behaviors as the general psychopathic population.

To date, there is no published evidence regarding the relationship between clinical outcomes, such as crime and recidivism data, and the specific group of low-anxious psychopaths identified using the PCL-R. Using data collected from Wisconsin state prisons, it is possible to examine the relationship between criminal versatility, number of violent crimes committed, number of non-violent crimes committed, and PCL-R psychopathy in 151 low-anxious and 163 high-anxious Caucasian inmates. Anxiety groups were determined using a median
split on the WAS and psychopathy groups were identified using recommended scores from the PCL-R manual (Hare, 1991). For these analyses, we dropped the item of criminal versatility and used prorated PCL-R scores.

To test the hypotheses, a multivariate analyses of variance (MANOVA) with PCL-R psychopathy (psychopaths, controls) and WAS anxiety (low, high) as the between-subjects variables was used. The dependent measures were criminal versatility, number of violent crimes committed, and number of nonviolent crimes committed. The results of the multivariate tests revealed a main effect for psychopathy ($F_{(5, 54.098, p < .001)}$) and a main effect for anxiety ($F_{(4.465, p < .01)}$). These main effects were qualified by a significant interaction between psychopathy and anxiety ($F_{(5.751, p < .01)}$). These main effects were qualified by a significant interaction between psychopathy and anxiety ($F_{(5.751, p < .01)}$). These main effects were qualified by a significant interaction between psychopathy and anxiety ($F_{(5.751, p < .01)}$).

As expected, the results of the between-subjects comparisons revealed that psychopathic individuals demonstrated more criminal versatility, $F(1, 310) = 194.135, p < .001$, more violent crimes, $F(1, 310) = 45.026, p < .001$, and more nonviolent crimes, $F(1, 310) = 67.849, p < .001$, than controls. For number of nonviolent crimes, there was also a significant main effect for anxiety, $F(1, 310) = 8.54, p < .01$, and significant psychopathy by anxiety interaction, $F(1, 310) = 11.538, p < .001$. The results indicated that low-anxious psychopathic individuals committed more offenses than low-anxious controls. No other main effects or interactions were significant.

Three planned comparisons were performed to test the specific hypothesis that low-anxious psychopathic individuals would commit more offenses (criminal versatility, violent, and nonviolent) than low-anxious controls. These comparisons were significant for criminal versatility, $t(102) = 8.51, p < 0.001$, number of violent crimes committed, $t(102) = 4.09, p = 0.001$, and number of nonviolent crimes committed, $t(102) = 6.99, p = 0.001$. As predicted, the low-anxious psychopathic individuals committed more offenses than low-anxious controls in every case (See Table 6).

The preliminary data, therefore, support the hypothesis that low-anxious psychopathic individuals demonstrate the same kinds of problematic antisocial behaviors as psychopaths more generally. This evidence, while limited, is consistent with the idea that the proposed Cleckley syndrome is associated with important clinical outcome data.

INTEGRATING THE TWO METHODS

Some readers may be confused by our recommendation of both the FFM and research investigating discrete bio-psychological mechanisms for parsing heterogeneity in psychopathy. Historically, psychologists researching discrete mechanisms have been considered at odds with proponents of the FFM and other models of general personality functioning. One reason for this is that the focus on bio-psychological mechanisms calls for the consideration of specific etiologies often viewed as simplistic by general personality theorists (Clark, 1998; Millon, 1991). From another perspective, proponents of discrete mechanisms have suggested that general personality theory is more descriptive and not as useful for explaining the root causes of problematic behavior.

Although these models generally represent different approaches to understanding psychopathology, it is our opinion that they need not be incompatible. In our discussion of mental retardation’s varied etiologies we have endeavored to explain how discrete bio-psychological mechanisms and variations in normal personality can exist side by side as different etiologies for the same

### Table 6. Criminal Activity in High- and Low-Anxious Psychopathic Individuals and Controls

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>No. Types of Crimes Committed M (SD)</th>
<th>No. Violent Crimes Committed M (SD)</th>
<th>No. Nonviolent crimes Committed M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-WAS Controls (n = 116)</td>
<td>4.34 (1.70)</td>
<td>2.31 (3.21)</td>
<td>10.77 (9.60)</td>
</tr>
<tr>
<td>High-WAS Controls (n = 95)</td>
<td>4.13 (1.83)</td>
<td>1.99 (2.46)</td>
<td>11.46 (11.18)</td>
</tr>
<tr>
<td>Low-WAS PP (n = 35)</td>
<td>7.54 (1.85)</td>
<td>6.03 (4.09)</td>
<td>27.85 (21.47)</td>
</tr>
<tr>
<td>High-WAS PP (n = 68)</td>
<td>7.07 (1.73)</td>
<td>5.56 (7.33)</td>
<td>18.57 (8.49)</td>
</tr>
</tbody>
</table>

Note. n, number of participants; M, mean; SD, standard deviation; WAS, Welsh Anxiety Scale; PP, psychopathic participants.
basic pathology. It is also possible that the two models, far from being incompatible, can be used in a complementary manner.

One means of doing this is to use the FFM to identify and specify individual variants of psychopathy with etiological significance. Research could then try to specify the bio-psychological processes underlying that specific etiology. In other words, the FFM could serve to identify the theoretically defined subgroups believed to manifest the kinds of mechanisms studied by laboratory researchers pursuing discrete mechanisms. Some examples of how this might be done have already been presented in our discussion of how the FFM might be used to help test theories of psychopathy presented by Lykken (1995), Patrick (1994), and Newman (1998). This approach might be particularly effective in integrating the search within the laboratory for discrete neurobiological or cognitive mechanisms with the basic science of individual differences research.

An additional method for integrating the two approaches has actually been suggested by Cleckley (1976). He proposed that the deficit underlying psychopathy might have different expressions that vary depending on the specific traits an individual possesses. He argued, for example, that criminality was not inherent to the definition of psychopathy, but noted that “when serious criminal tendencies do emerge in the psychopath, they gain ready expression” (Cleckley, 1976, p. 262). The implication, then, is that the etiology makes psychopathic individuals disinhibited and that behaviors towards which they might otherwise be inclined become excessive as a result.

Following this logic, a potentially productive course of study would be to examine how the presence of different FFM traits impacts the expression of a specific bio-psychological deficit such as poor response modulation. For example, we might expect that an individual with poor response modulation and a high antagonism score would be more likely to display disinhibited aggression than one low in antagonism. Systematic study of the effect different FFM profiles have on the expression of key bio-psychological deficits may prove to be a powerful way to help explain observed variations in the display of psychopathic behavior.

To reiterate, we believe that the FFM and the study of discrete bio-psychological mechanisms provide different, but potentially compatible, perspectives on understanding psychopathology. Although researchers often focus on the differences between these two models, we believe there is more to be gained by searching for means of integrating them. Each model provides different strengths that may help us categorize, describe, and study the various etiologies that are potentially associated with psychopathy.

**CONCLUSIONS**

In this manuscript we have argued that psychopathy as it is currently diagnosed identifies a heterogeneous collection of repeat offenders who may not share a common etiology or pathology. Psychologists will need to understand the etiological processes underlying these different forms of psychopathy in order to develop effective clinical interventions. We believe that developing an understanding of these various etiologies will require researchers to use both the PCL-R and other measures to identify theoretically homogeneous groups for study. Towards this end, we have presented the FFM and the response-modulation hypothesis as examples of how theory can be used to help refine classification of psychopathic variants and generate hypotheses about the etiological processes at work in these groups.

**NOTES**

1. Despite the impressive evidence supporting the validity of the FFM, there have been some influential critiques of the model; for example, see Block (1995) and Loevinger (1994).

2. Loevinger (1994) has argued that the FFM’s Conscientiousness is primarily a measure of conformity and neglects advanced stages of ego/moral development. To the extent that this is true, it may impact the degree to which the FFM profile measures Cleckley’s (1976) psychopathy vs idealistic non-conformity. Preliminary correlations with other measures of antisocial behavior/psychopathy are, however, reassuring (Lynam 2002; Miller & Lynam, 2001; Miller et al., 2001).

3. To create the Widiger and Lynam prototype, we scored facets hypothesized to be negatively related to psychopathy as 1 and facets hypothesized to be positively related as 5; facets not used in the description were given scores of 3 (see Miller et al., 2001 for more details).

4. Although there are distinctions between anxiety and fear (Barlow, Chorpita, & Turovsky 1996), it has been argued that both fall under a general negative affect domain at a trait level (Watson & Clark, 1992). Readers should be aware, however,
that research with Tellegen’s model has suggested that these traits load on different factors (fearfulness on Constraint and anxiety on Negative Emotionality; White & Depue, 1999).

5. Although Newman (1998) has made attempts to explain various aspects of psychopathy in terms of his theory, some critics have remained skeptical that the observed range of affective-motivation pathology could be explained by what is essentially an attention deficit (Hare, 1998; Lykken, 1995).

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